UNITED STATES PATENT AND TRADEMARK OFFICE

UNITED STATES DEPARTMENT OF COMMERCE
United States Patent and Trademark Office
Address: COMMISSIONER FOR PATENTS
P.O. Box 1450
Alexandria, Virginia 22313-1450
www.uspto.gov

APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/581,431	02/08/2008	Carlos F Barbas	TSRI 1019. 1 PCT	5101

39843 7590 09/16/2011

BELL & ASSOCIATES
58 West Portal Avenue No. 121
SAN FRANCISCO, CA 94127

EXAMINER	
HADDAD, MAHER M	
ART UNIT	PAPER NUMBER
1644	

NOTIFICATION DATE	DELIVERY MODE
09/16/2011	ELECTRONIC

Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Notice of the Office communication was sent electronically on above-indicated "Notification Date" to the following e-mail address(es):

abell@bell-iplaw.com
mkaser@bell-iplaw.com
info@bell-iplaw.com

Office Action Summary	Application No.	Applicant(s)	
	10/581,431	BARBAS ET AL.	
	Examiner	Art Unit	
	MAHER HADDAD	1644	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *08 July 2011*.

2a)☒ This action is **FINAL**. 2b)☐ This action is non-final.

3)☐ An election was made by the applicant in response to a restriction requirement set forth during the interview on _____; the restriction requirement and election have been incorporated into this action.

4)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

5)☒ Claim(s) *1-32* is/are pending in the application.

5a) Of the above claim(s) *1-4,11-14,17-21,24,27 and 30-32* is/are withdrawn from consideration.

6)☐ Claim(s) _____ is/are allowed.

7)☒ Claim(s) *5-10, 15-16, 22-23, 25-26, and 28-29* is/are rejected.

8)☐ Claim(s) _____ is/are objected to.

9)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

10)☐ The specification is objected to by the Examiner.

11)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

12)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

13)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

RESPONSE TO APPLICANT'S AMENDMENT

1. Applicant's amendment, filed 07/08/2011, is acknowledged.

2. Claims 1-32 are pending.

3. Claims 1-4, 11-14, 17-21, 24, 27, and 30-32 stand withdrawn from further consideration by the Examiner, 37 C.F.R. § 1.142(b) as being drawn to a nonelected invention.

4. Claims 5-10, 15-16, 22-23, 25-26, and 28-29 are under consideration in the instant application as they read on an antibody that specifically immunoreacts with integrin $\alpha_{IIB}\beta 3$.

5. Applicant is advised that should claim 8 be found allowable, claim 10 will be objected to under 37 CFR 1.75 as being a substantial duplicate thereof. When two claims in an application are duplicates or else are so close in content that they both cover the same thing, despite a slight difference in wording, it is proper after allowing one claim to object to the other as being a substantial duplicate of the allowed claim. See MPEP § 706.03(k).

6. In view of the amendment filed on 07/08/2011, only the following rejections are remained.

7. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

8. Claims 5-10, 15-16, 22-23, 25-26, and 28-29 stand rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Applicant is in possession of an anti-αIIBβ3 antibody comprising VH of SEQ ID NOs: 32-38 or RAD3 (VH SEQ ID NO: 36), RAD4, RAD9 (VH SEQ ID NO: 33), RAD11, RAD12 (VH SEQ ID NO: 34), RAD32 (VH SEQ ID NO: 37), RAD34 (VH SEQ ID NO: 35), RAD87 (VH SEQ ID NO: 32) and RAD88 (VH SEQ ID NO: 38) (once the deposit is satisfied), wherein the antibody comprises a heavy chain CDR3 motif Arg-Ala-Asp (RAD).

Applicant is not in possession of the protein claimed in claims 5-10, 15-16, 22-23, 25-26, and 28-29 for the same reasons set forth in the previous Office Action mailed 01/12/2011.

Applicant's arguments, filed 07/08/2011, have been fully considered, but have not been found convincing.

Applicants submit that the claimed antibodies are disclosed in the specification at page 23, lines 25-31, at page 24, lines 17-27, at page 25, lines 3-21, at page 26, lines 33-35, at page 27, lines 1-18, Figure 1, Figure 5, and Table 3, where Applicants disclosed the antibody as claimed was specific for integrin αIIBβ3 and that cyclic linear peptides having identical sequences to the antibodies assayed had similar inhibitory activity, thereby confirming that the particular amino acid sequence was the active moiety in the immunoreaction between antibody and integrin αIIBβ3. The use of the specific inhibitory peptides indicates that the particular amino acid sequence in the CDR3 region is involved with integrin αIIBβ3 binding and that the other CDRs, as suggested by the Examiner's reading of the prior art, do not need to be defined. The claimed invention overcomes the prior art problems and the improvements over the prior art are discussed in the specification at pages 27 through 31.

Applicants note that the method for using either light chain or heavy chain immunoglobulins are disclosed in the specification at page 9, last paragraph where a phage display assay is disclosed. Methods in more detail are disclosed at page 10, first paragraph and at page l 1, second paragraph where Applicants stated that "antibody molecules having identical, or functionally equivalent, amino acid residue sequences in their CDR regions have the same binding specificity". In addition, at page 10, third paragraph where Applicants specifically stated "it is also possible to determine, without undue experimentation, if a human monoclonal antibody has the same (i.e., equivalent) specificity as a human monoclonal antibody of this invention". As discussed above, the use of the specific inhibitory peptides indicates that the particular amino acid sequence in the CDR3 region is involved with integrin αIIBβ3 binding and that the other CDRs, as suggested by the Examiner's reading of the prior art, do not need to be defined. The claimed invention overcomes the prior art problems and the improvements over the prior art are discussed in the specification at pages 27-31.

This is not found to be persuasive because the scope of the claims encompasses any antibody comprising HCDR3 of SEQ ID NOS: 8 and 25-31 or a protein comprising tripeptide motif RAD would bind to αIIBβ3 integrin. Neither the specification, nor the prior art methods result in an antibody solely by keeping HCDR3 in the VH defined and randomizing the rest of the VH and VL domains. Moreover, neither the specification, nor the prior art provides any examples to support the premise that HCDR3 of the VH/VL solely responsible for antigen binding within an antibody. The prior art does not support a definition of an antibody structure solely by defining the HCDR3 sequence of a VH. The specification fails to show that a single HCDR3 specificity of the anti-αIIBβ3 antibody influences the specificity for any antigen binding antibody. The specification fails to establish that by replacing HCDR3 of any antibody (e.g., αIIBβ3, αvβ3, αvβ3, α5β1, CD30 or CD47) with amino acid of SEQ ID NO: 8 and 25-31 lead to αIIBβ3 integrin selectivity switch in concert with the HCDR3 replacement. Substituting the HCDR3 of SEQ ID NOs: 8 and 25-31 in an antibody has not been shown to lead to αIIBβ3 integrin selectivity switch in concert with the substituted HCDR3. Such teachings is not part of the prior art and were not made part of the specification at the time the invention was made. The instant specification fails to provide sufficient direction or guidance to make the claimed genus of anti-αIIBβ3 antibodies. Note that the Examiner's position is consistent with Office policy (http://www.cabic.com/bcp/) as put forth in the presentation of Bennett Celsa entitled "Written

Description: Antibodies", which describes how claims directed to defined HCRD3 sequences fail to meet the requirements under 35 USC 112, first paragraph for written description. In accordance with these concepts, Example 3 of the Written description Training Materials (slide 42-47) explains that, in the context of claiming an antibody comprising HCDR3 recited a claim lacks descriptive support because neither the specification, nor the prior art provides any examples to support the premise that CDR3 of the VH or VL is solely responsible for antigen binding. Prior art does not support a definition of an antibody structure solely by defining the CDR3 sequence of a VH or VL.

For further support, the Examiner points to Simmons et al (Proteins 2008; 71:119–130) teachings that crystallographic studies of recombinant proteins 1-A-7 and 1-A-11 showed that the SYP motifs on these VNARs presented at the tip of the exposed CDR3 loops, ideally positioned within bulge-like structures to make contact with the MAb5G8 antibody. These loops, in particular in 1-A-11, were further stabilized by inter- and intra- loop disulphide bridges, hydrogen bonds, electrostatic interactions, and aromatic residue packing. We rationalize the higher affinity of the VNARs compared to the parental antigen by suggesting that adjacent CDR1 and framework residues contribute to binding affinity, through interactions with other CDR regions on the antibody, though of course definitive support of this hypothesis will rely on co-crystallographic studies (see abstract).

9. Claims 5-10, 15-16, 22-23, 25-26, and 28-29 stand rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for an anti-αIIBβ3 antibody comprising VH of SEQ ID NOs: 32-38 or RAD3 (VH SEQ ID NO: 36), RAD4, RAD9 (VH SEQ ID NO: 33), RAD11, RAD12 (VH SEQ ID NO: 34), RAD32 (VH SEQ ID NO: 37), RAD34 (VH SEQ ID NO: 35), RAD87 (VH SEQ ID NO: 32) and RAD88 (VH SEQ ID NO: 38) (once the deposit is satisfied), wherein the antibody comprises a heavy chain CDR3 motif Arg-Ala-Asp (RAD), does not reasonably provide enablement for the antibodies claimed in claims 5-10, 15-16, 22-23, 25-26, and 28-29. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims for the same reasons set forth in the previous Office Action mailed 01/12/2011.

Applicant's arguments, filed 07/08/2011, have been fully considered, but have not been found convincing.

With respect to the pharmaceutical composition, Applicants point that the first sentence of the Background of the Invention recites "integrin αIIBβ3 inhibitors are new class of antithrombotic agents that block fibrinogen binding to the platelet integrin αIIBβ3, thereby inhibiting platelet-platelet interactions essential for the formation of platelet thrombi" (see page 1, lines 7-9). Applicants therefore submit that inhibition of fibrinogen binding to platelet integrin αIIBβ3 is well known by those of skill in the art to block and treat thrombus formation (thrombosis).

Furthermore, applicants particularly disclosed that antibodies and antibody Fab fragments can be used to in vivo or in vitro modulate the function of integrin αIIBβ3 on platelets (page 16, lines 12-14 and 29-30), is capable of inhibiting the aggregation of platelets, and thereby decreasing the rate of thrombus formation (page 16, lines 17-18 and 30-31), and pages 16-18 throughout. Applicants respectfully submit that such experiments clearly may be used by those of skill in the art to predict how the antibody can be used to treat formation of thrombi in vivo. Applicants submit that such experiments are commonly used to determine the potential formation of thrombi in clinical samples and therefore one of skill in the relevant art would appreciate that such results are of objective clinical relevance.

However, regarding the intended *in vivo* methods which rely on generally unpredictable mechanisms, "The amount of guidance or direction needed to enable the invention is inversely related to the amount of knowledge in the state of the art as well as the predictability in the art." In re Fisher, 427 F.2d 833, 839, 166 USPQ 18, 24 (CCPA 1970). The "amount of guidance or direction" refers to that information in the application, as originally filed, that teaches exactly how to make or use the invention. The more that is known in the prior art about the nature of the invention, how to make, and how to use the invention, and the more predictable the art is, the less information needs to be explicitly stated in the specification. In contrast, if little is known in the prior art about the nature of the invention and the art is unpredictable, the specification would need more detail as to how to make and use the invention in order to be enabling (MPEP 2164.03)." The MPEP also states that physiological activity can be considered inherently unpredictable.

However, the instant claims are drawn to a large genus for the use of methods which have not been developed yet to the point where a specific benefit exists in currently available form. Given the relatively incomplete understanding in correlating in vitro assays and in vivo animal models to clinical treatment of platelet thrombi involved, and the lack of a reasonable correlation between the narrow disclosure in the specification and the broad scope of protection sought in the claims, the claims are not enabled. See MPEP 2164.08. There must be a rigorous correlation of pharmacological activity between the disclosed in vitro utility and an in vivo utility to establish practical utility.

Applicants submit that claim 5 recites an antibody comprising an amino acid residue sequence selected from the group consisting of SEQ ID Nos: 8, 25, 26, 27, 28, 29, 30, and 31. Applicants submit that the antibody specifically immunoreacts with integrin αIIBβ3. Applicants submit that such antibodies are disclosed in the specification at page 23, lines 25-31, at page 24, lines 17-27, at page 25, lines 3-2 I, at page 26, lines 33-35, at page 27, lines 1-18, Figure 1, Figure 5, and Table 3, where Applicants disclosed the antibody as claimed was specific for integrin αIIBβ3 and that cyclic linear peptides having identical sequences to the antibodies assayed had similar inhibitory activity, thereby confirming that the particular amino acid sequence was the active moiety in the immunoreaction between antibody and integrin αIIBβ3. The use of the specific

inhibitory peptides indicates that the particular amino acid sequence in the CDR3 region is involved with integrin αIIBβ3 binding and that the other CDRs, as suggested by the Examiner's reading of the prior art, do not need to be defined. The claimed invention overcomes the prior art problems and the improvements over the prior art are discussed in the specification at pages 27 through 31.

This is not found to be persuasive because the scope of the claims encompasses any antibody comprising HCDR3 of SEQ ID NOS: 8 and 25-31 or a protein comprising tripeptide motif RAD would bind to αIIBβ3 integrin. Neither the specification, nor the prior art methods result in an antibody solely by keeping RAD/HCDR3 in the VH defined and randomizing the rest of the VH and VL domains. Moreover, neither the specification, nor the prior art provides any examples to support the premise that HCDR3 of the VH/VL solely responsible for antigen binding within an antibody. The prior art does not support a definition of an antibody structure solely by defining the HCDR3 sequence of a VH. The specification fails to show that a single HCDR3 specificity of the anti-αIIBβ3 antibody influences the specificity for any antigen binding antibody. The specification fails to establish that by replacing HCDR3 of any antibody (e.g., αIIBβ3, αvβ3, αvβ3, α5β1, CD30 or CD47) with amino acid of SEQ ID NO: 8 and 25-31 lead to αIIBβ3 integrin selectivity switch in concert with the HCDR3 replacement. Substituting the HCDR3 of SEQ ID NOs: 8 and 25-31 or RAD motif in an antibody has not been shown to lead to αIIBβ3 integrin selectivity switch in concert with the substituted HCDR3. Such teachings is not part of the prior art and were not made part of the specification at the time the invention was made. The instant specification fails to provide sufficient direction or guidance to make the claimed genus of anti- αIIBβ3 antibodies. For support, the Examiner points to Simmons et al (Proteins 2008; 71:119–130) teachings that crystallographic studies of recombinant proteins 1-A-7 and 1-A-11 showed that the SYP motifs on these VNARs presented at the tip of the exposed CDR3 loops, ideally positioned within bulge-like structures to make contact with the MAb5G8 antibody. These loops, in particular in 1-A-11, were further stabilized by inter- and intra- loop disulphide bridges, hydrogen bonds, electrostatic interactions, and aromatic residue packing. We rationalize the higher affinity of the VNARs compared to the parental antigen by suggesting that adjacent CDR1 and framework residues contribute to binding affinity, through interactions with other CDR regions on the antibody, though of course definitive support of this hypothesis will rely on co-crystallographic studies (see abstract).

Note that the Examiner's position is consistent with Office policy (http://www.cabic.com/bcp/) as put forth in the presentation of Larry Helms entitled "Enablement Issues in the Examination of Antibodies", which describes how claims directed to defined HCRD3 sequences fail to meet the requirements under 35 USC 112, first paragraph for enablement. In accordance with these concepts, Example 3 of the enablement Training Materials (slide 16-28) explains that, in the context of claiming an antibody comprising HCDR3 recited a claim lacks enablement because the prior art does not show screening for antibodies by just defining CDR3. The methods rely on using an entire VH or VL and screening random complimentary chains. The prior art does not show that a CDR3 is universally solely responsible for antigen binding. The prior art does not support a definition of an antibody structure solely by defining the CDR3 sequence of a VH.

Applicants further note that the method for using either light chain or heavy chain immunoglobulins are disclosed in the specification at page 9, last paragraph where a phage display assay is disclosed. Methods in more detail are disclosed at page 10, first paragraph and at page 11, second paragraph where Applicants stated that "antibody molecules having identical, or functionally equivalent, amino acid residue sequences in their CDR regions have the same binding specificity". In addition, at page 10, third paragraph where Applicants specifically stated "it is also possible to determine, without undue experimentation, if a human monoclonal antibody has the same (i.e., equivalent) specificity as a human monoclonal antibody of this invention". Therefore, Applicants submit that the present application does describe in such a clear and sufficient manner as to enable those skilled in the art to practice the invention.

However, the Examiner does not agree that the instant specification provides sufficient direction or guidance to make the claimed genus of antibodies and that no more than routine experimentation would be required to make the claimed genus of antibodies. The claims are drawn to antibodies comprising only HCDR3 alone. The scope of the claim encompasses antibodies with HCDR3 in the place of HCDR1 or HCDR2, LCDR1-3 as well as a subgenus of antibodies that encompass only HCDR3. Neither the specification, nor the prior art does not support a definition of an antibody structure solely by defining the CDR3 sequence of a VH. While it is ture that the Office acknowledges "skilled artisans can screen libraries to identify other variable domains that will pair with the starting variable domain and maintain antigen specificity" this is not the same thing as making the genus of anti- αIIBβ3 antibodies containing a particular CDR3 in the context of VH or VL and framework regions capable of binding αIIBβ3 with low affinity and efficaciously treat thrombosis.

10. The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

A person shall be entitled to a patent unless --

(b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

(e) the invention was described in a patent granted on an application for patent by another filed in the United States before the invention thereof by the applicant for patent, or on an international application by another who has fulfilled the requirements of paragraphs (1), (2), and (4) of section 371(c) of this title before the invention thereof by the applicant for patent.

The changes made to 35 U.S.C. 102(e) by the American Inventors Protection Act of 1999 (AIPA) and the Intellectual Property and High Technology Technical Amendments Act of 2002 do not apply when the reference is a U.S. patent resulting directly or indirectly from an international application filed before November 29, 2000. Therefore, the prior art date of the reference is determined under 35 U.S.C. 102(e) prior to the amendment by the AIPA (pre-AIPA 35 U.S.C. 102(e)).

11. Claims 15, 23, 26 and 29 stand rejected under 35 U.S.C. 102(e) as being anticipated by US Patent 7,812,136 B2 for the same reasons set forth in the previous Office Action mailed 01/12/2011.

Applicant's arguments, filed 07/08/2011, have been fully considered, but have not been found convincing.

Applicant submits that VL and VL of SEQ ID NO:159 does not disclose tripeptide sequence RAD (ARg-Ala-Asp) nor do any of the claimed SEQ ID NOs: 8 and 25-31.

However, it appears that applicant is reading the limitation of dependent claim 16 into the base claim 15. The scope of the antibody recited in base claim 15 does not require the presence of the tripeptide RAD motif in the anti-αIIBβ3 antibodies, rather the claimed antibodies have only to compete with binding activity of another protein comprising RAD. The referenced GPIIb protein comprises RAD and would compete for binding of the reference antibody in a standard completion assay in the absence of evidence to the contrary.

12. Claims 15, 23, 26 and 29 stand rejected under 35 U.S.C. 102(b) as being anticipated by Quinn et al (*Circulation.* 1999;99:2231-2238.) for the same reasons set forth in the previous Office Action mailed 01/12/2011.

Applicant's arguments, filed 07/08/2011, have been fully considered, but have not been found convincing.

Applicant submits that Quinn et al. do not teach nor suggest an antibody comprising SEQ ID NOs: 8, 25, 26, 27, 28, 29, 30, and 31 as recited in base claim l, upon which claim 10 depends. Applicant submits that claim 10 has all the limitations of base claim 5 and the Examiner has not shown where Quinn et al. teach all the limitations of claim 5. Applicant submits that the prior art and the subject matter of the claims at issue are different and therefore that Quinn et al. do not anticipate claims 10, 15-16, 23, 26, and 29.

However, the scope of the antibody recited in base claim 15 does not require the presence of the tripeptide RAD motif in the anti-αIIBβ3 antibodies, rather the claimed antibodies have only to compete with binding activity of another protein comprising RAD. The referenced GPIIb protein comprises RAD and would compete for binding of the reference antibody in a standard completion assay in the absence of evidence to the contrary.

13. Biris et al reference cited on the PTO-892 because it is art of interest. Biris et al teach an antibody that binds to RAD adhesive motifs, encompassed in αIIb 313–332.

14. No claim is allowed.

15. **THIS ACTION IS MADE FINAL.** Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the mailing date of this final action.

16. Any inquiry concerning this communication or earlier communications from the examiner should be directed to Maher Haddad whose telephone number is (571) 272-0845. The examiner can normally be reached Monday through Friday from 7:30 am to 4:00 pm. A message may be left on the examiner's voice mail service. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Ram Shukla can be reached on (571) 272-0735. The fax number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

September 8, 2011

/Maher M. Haddad/
Primary Examiner,
Art Unit 1644